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Salivary Beta Defensins 1 in Relation to Dental Caries

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Abstract

Objective: This study aimed to investigate an association between caries susceptibility among adolescents in Baghdad city and variants in beta defensins 1 (*DEFB1*). **Materials and Methods:** This study was conducted among 78 adolescents (39 with very low caries experience and 39 with high caries experience) aged 13–15 years old from Baghdad city. Two single nucleotide polymorphisms (SNPs) were identified from unstimulated saliva samples, namely rs2738182 and rs1800972. Dental caries was evaluated with the DMFS/ DMFT (decayed, missing, filled surface/teeth) and D1-4 indices. **Results:** None of the studied SNPs revealed significant difference between the high and very low caries groups. **Conclusion:** Lack of relationship between *DEFB1* polymorphisms and susceptibility to caries development underscore the complexity of genetic influences on caries susceptibility and the importance of considering different genetic and environmental factors.

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Introduction

Cariology is a deeply intricate branch of dental research, largely because dental caries—the most common infectious oral disease globally—is a multifactorial condition. Its development isn't caused by a single trigger; rather, it emerges from a complex interplay between an individual's genetics, lifestyle, and socioeconomic status, alongside the specific biological characteristics of their oral environment [1-3]. In spite the development of techniques to prevent and treat it, dental caries still concerns major problem which affect quality of life [4,5]. The progression of dental caries is a time-dependent process that occurs when a susceptible host is consistently exposed to cariogenic bacteria and fermentable carbohydrates.

Beyond this primary interaction, the disease is heavily moderated by specific host-related biological factors. These include the protective qualities of saliva—specifically its flow rate and buffering capacity—as well as physical dental traits like tooth alignment, the structural integrity of the enamel surface, and the specific morphology (depth) of occlusal fissures [3,6].

Saliva serves as the body's natural protective mechanism, offering a range of innate and acquired defense factors that can effectively hinder bacterial invasion, growth, and metabolism. These protective mechanisms operate through various means, including inhibiting bacterial adherence and the production of acids by *streptococci*. Modern proteomics and peptideomics have identified a diverse array of non-immunoglobulin antimicrobial proteins (AMPs) that serve as the primary line of innate defense [7,8].

Salivary biomarkers have emerged as a useful diagnostic tool to assess both oral and systemic health. These biomarkers can detect qualitative changes that signify variations in the oral environment, enabling the identification of participants with an elevated susceptibility to oral diseases, pinpointing sites with ongoing disease activity, predicting future sites at risk of deterioration, and monitoring the effectiveness of therapeutic interventions, this approach can contribute to more personalized and effective management of oral health [9].

Defensins are known as antimicrobial peptides that are rich with lysine and arginine amino acids in their structure. Arginine in saliva could be derived from defensins and other arginine-rich peptide cleavage. Arginine and its related form shown to have a protective role against dental

caries. Suggesting that both beta-defensin and alpha-defensin reduce possibilities for caries experience and are connected to absence of caries [10].

The *DEFB1* gene located at 8p22–23 and encodes for the hBD1 antimicrobial peptide which is expressed consistently. It is considered a promising candidate for investigating genetic susceptibility to dental caries. Genetic variations in this gene may potentially disrupt the production of hBD1 which is a key player in defending the oral mucosa. Such disruptions could result in an increased susceptibility to oral pathogen infections and may also contribute to the development of dental caries and periodontitis [11].

No previous studies in Iraq have linked genes of antimicrobial elements, such as beta defensin 1 (*DEFB1*) in saliva, with dental caries. Therefore, the current study aimed to identify and screen susceptible patients, and to understand the contribution of genes in the pathogenesis of caries. As DEFB1 is localized in the oral cavity, we conducted tests to investigate if variations in the *DEFB1* gene are associated with caries.

Materials and Methods

This eight-month study was conducted at a specialized dental center in Al-Kadhimiya, Baghdad, involving a sample of patients attending the facility. Prior to participation, informed consent was secured from the guardians of all subjects. The research focused on 78 unrelated individuals, categorized into two equal groups: 39 with very low caries and 39 with high caries. Clinical procedures included the collection of unstimulated saliva, oral examinations and followed by DNA analysis to investigate two specific single-nucleotide polymorphisms (SNPs) within *DEFB1*.

Patients within the age range of 13-15 years old regardless of gender were included. They were Iraqi Arab Population with no history of any systemic diseases or medications in the preceding three weeks. They had either high caries scores with DMFT more than 9, and low caries scores persons with DMFT less than 2 [12].

Patients with any systemic disease, clefts, congenital anomalies, generalized dental problems or who wore a fixed orthodontic appliance were not included in this study. Patients that received fluoride supplements or had fissure sealants were also not included. Subjects with DMFT more than 2 but less than 9 were not included in the current study.

At first, unstimulated saliva collected from each participant then oral examinations were done, saliva used to extract DNA which was being centrifuged at 12000 revolution per minute (rpm) for 3 minutes. Clinical examination was conducted using disposable mouth mirror and dental explorer after drying of the field of examination with air triple. Dental caries was evaluated with the DMFT/DMFS (decayed, missing, filled teeth) index, for each patient as recommended by World Health Organization Guidelines Severity of dental and caries according to criteria of WHO (1979) [13].

Genomic DNA was isolated from saliva sample according to the protocol ReliaPrep™ Saliva gDNA Miniprep System, Promega. Quantus Fluorometer was used to detect the concentration of extracted DNA to detect the quality of samples for downstream applications.

PCR primers were supplied by Macrogen Company in a lyophilized form. The DNA template was amplified with the same primer pair, (Forward) (Reverse). After PCR amplification, agarose gel electrophoresis was adopted to confirm the presence of amplification. PCR was completely dependable on the extracted DNA criteria.

PCR products were sent for Sanger sequencing using ABI3730XL, automated DNA sequencer by Macrogen Corporation – Korea. The results were received by email then analyzed using geneious software.

Computerized software statistical package for social science (SPSS version-24) was used. The variation of frequencies between groups were analysed using chi-square test. Hardy-Weinberg equilibrium (HWE) was used to calculate the expected common homozygotes, expected heterozygotes, expected rare homozygotes. Chi-square test was used to find out genotype deviation from HWE, and to compare the distributions of genotypes and allele frequencies in the disease and control groups. The relative risk (RR) is the real measure of association between exposure to a certain factor and having the disease or outcome. The risk associated with individual genotypes or alleles was calculated as the odds ratio (OR) with 95% confidence intervals (95% CI). Which indicate how many times more frequently a disease develops in individuals carrying the allele or genotype than in individuals lacking it.

The study was conducted in accordance with the ethical principles that had their origin in the Declaration of Helsinki. It was carried out with patients verbal and analytical approval before sample was taken. The study protocol and the subject information and consent form were reviewed and approved by a local ethics committee according to the document number 366 (including the number and the date in 1/8/2021) to get this approval.

Results

Sequencing of *DEFB1* in the present study resulted in detection of two SNPs (rs2738182, rs1800972). rs2738182 is a substitution of thymine (T) for an adenine (A) while rs1800972 had substitution of guanine (G) with cytosine (C).

The genotype and allele frequency comparisons calculated for *DEFB1* SNPs are presented in Tables 1 and 2. There was no significant differences in allele or genotype frequencies.

Discussion

The growing interest in understanding the factors contributing to an individual's susceptibility to caries aligns with the advancement of viable methods for comprehending genetic susceptibility to complex human diseases, particularly the tools are stemming from the Human Genome Project. Factors like tooth structure, buffering capacity, salivary flow, dietary habits, oral microbiota, oral care routines and a history of prior dental caries all significantly affect the development of carious lesions [14,15]. However, compelling evidence suggests that an individual's genetic makeup can indeed influence their predisposition to dental caries [16,17].

None of the studied SNPs revealed significant difference between the high and very low caries groups. In addition, there are no significant correlations among genotypes of each SNPs in both groups with caries severity. This lack of relationship between *DEFB1* polymorphisms (rs1800972) and susceptibility to caries development agreed with a sample of Turkish children has been documented [18]. These variations in study populations and findings underscore the complexity of genetic influences on caries susceptibility and the importance of considering different genetic and environmental factors.

Another explanation to understand this study findings' is context of a multifactorial disease as dental caries are a complex disease. While innate immunity has been linked to caries susceptibility, it is only one component of the picture. Other factors, like as genetic polymorphisms in genes involved in enamel development and others have been related to a higher vulnerability to caries. Recognizing caries' multidimensional nature allows better understand and treat this prevalent dental illness [18].

As for the genetic variations mentioned, rs2738182 are not reported in ClinVar, a database of genetic variants and their clinical significance. This study was the first study which evaluated these SNPs and established clinical associations with dental caries at this time.

Further research may be needed to understand their potential implications.

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Table 1. Genotypes and alleles frequency comparisons calculated for rs2738182.

rs2738182 (T/A)		High caries n=39	Very low caries n=39	OR	CI	RR	χ2	*P*-value	Alleles frequency	High caries n=39	Very low caries n=39	χ2	*P*-value
Homozygous	TT	7 (17.95%)	11 (28.2%)	0.56	(0.19-1.68)	0.64	0.889	0.345	T	36 (46.1%)	44 (56.4 %)	1.642	0.2
Heterozygous	TA	22 (56.4%)	22 (56.4%)	1.0	(0.40-2.44)	1.57	0.00	1.0	A	42 (53.8%)	34 (43.5%)		
Homozygous	AA	10 (25.6%)	6 (15.3%)	1.90	(0.61-5.88)	1.67	1.00	0.317					
HWE χ^2		0.709	0.843						HWE *P*-value	0.399	0.358		
P-value		0.701	0.655										

Abbreviations: OR: Odd Ratio; RR: Relative Risk; X2: Chi-Square test; HWE: Hardy-Weinberg Equilibrium; P- value probability value; NS non-significant.

Table 2. Genotypes and alleles frequency comparisons calculated for rs1800972.

rs1800972 (G/C) Genotypes frequency		High caries n=39	Very low caries n=39	OR	CI	RR	χ2	*P*-value	alleles frequency	High caries n=39	Very low caries n=39	χ2	*P*-value
Homozygous	GG	1 (2.56%)	1 (2.56%)	1.0	(0.06-16.5)	1.0	0.00	1.00 NS	G	9 (11.5%)	12 (15.3 %)	0.495	0.481
Heterozygous	GC	7 (17.95%)	10 (25.6%)	0.63	(0.21-1.88)	0.7	0.529	0.466	C	69 (88.4%)	66 (84.46%)		
Homozygous	CC	31 (79.4%)	28 (71.7%)	1.52	(0.53-4.32)	1.1	0.152	0.696					
HWE χ^2		0.568	0.008						HWE *P*-value	0.45	0.924		
P-value		0.752	0.995										

Abbreviations: OR: Odd Ratio; RR: Relative Risk; X2: Chi-Square test; HWE: Hardy-Weinberg Equilibrium; P- value probability value; NS non-significant.